



Communicating Research in Clear Language

The Oxytocin Receptor and Social Abilities Across Neurodevelopmental Disorders

What is the research about?

Oxytocin, a chemical produced in the brain's pituitary gland, is known to influence social behaviour. Its receptor, OXTR, is a region on a cell that is sensitive to oxytocin, and can initiate changes in a cell's activity, ultimately altering social behaviour in an individual. There are multiple variations (called single nucleotide polymorphisms or SNPs) in the OXTR gene that are known to be related to differences in the social abilities of typically developing populations. However, it is not known if the same response is found in children with neurodevelopmental disorders, most of whom have some degree of social skills deficits. This study looked at what effects, if any, variations in the OXTR gene had on the social abilities of children with ASD or ADHD.

What did the researchers do?

The researchers first assessed the social skills of 341 children with Autism Spectrum Disorder (ASD) and 276 children with Attention Deficit/Hyperactivity Disorder (ADHD). They then compared the social skills by diagnosis and by different OXTR gene variants.

What did the researchers find?

The findings were unexpected. The two OXTR gene variants best studied in the general population were associated with the increased severity of social behaviour deficits in children with ASD. However, these same OXTR gene variants appeared to produce lower levels of social skills deficits in children with ADHD. Thus, variation in the OXTR gene appears to affect social skills deficits differently depending on the underlying disorder.

Take home message

This study of variations of the oxytocin receptor gene, OXTR, in children with ASD or ADHD, produced unexpected outcomes. The two most common variants of OXTR were associated with greater severity of social skills deficits in children with ASD, while the same variants were associated with reduced levels of social skills deficits in children with ADHD. The authors call for studies of these variants at their basic, molecular level to attempt to discover the reason for this inconsistency.

NOTE: The original [Research Report](#) was written by D. A. Baribeau and colleagues, and was published in *Scientific Reports*. 2017.